

FILE 'MEDLINE' ENTERED AT 14:44:53 ON 18 JUL 2002

```

L1      19214 S NEMATODE OR C.ELEGANS OR ?ELEGANS
L2      509 S PROLYL ?HYDROXYLASE
L3      2 S L1 (S) L2
L4      2 S L1 (L) L2
L5      7 S DPY-18
L6      103 S DPY-18 OR DPY
L7      59 S L1 (L) L6
L8      0 S L7 AND L2

```

=> FILE CAPLUS MEDLINE BIOSIS EMBASE  
COST IN U.S. DOLLARS

SINCE FILE ENTRY	TOTAL SESSION
3.06	3.27

FULL ESTIMATED COST

FILE 'CAPLUS' ENTERED AT 14:49:21 ON 18 JUL 2002  
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.  
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.  
COPYRIGHT (C) 2002 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'MEDLINE' ENTERED AT 14:49:21 ON 18 JUL 2002

FILE 'BIOSIS' ENTERED AT 14:49:21 ON 18 JUL 2002  
COPYRIGHT (C) 2002 BIOLOGICAL ABSTRACTS INC.(R)

FILE 'EMBASE' ENTERED AT 14:49:21 ON 18 JUL 2002  
COPYRIGHT (C) 2002 Elsevier Science B.V. All rights reserved.

=> S L3  
L9 11 L3

=> DUP REM L9  
PROCESSING COMPLETED FOR L9  
L10 6 DUP REM L9 (5 DUPLICATES REMOVED)

=> D 1-6 IBIB ABS

L10 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 1

ACCESSION NUMBER: 2001:757463 CAPLUS

DOCUMENT NUMBER: 136:33452

TITLE: C. elegans EGL-9 and mammalian homologs define a family of dioxygenases that regulate HIF by prolyl hydroxylation

AUTHOR(S): Epstein, Andrew C. R.; Gleadle, Jonathan M.; McNeill, Luke A.; Hewitson, Kirsty S.; O'Rourke, John; Mole, David R.; Mukherji, Mridul; Metzen, Eric; Wilson, Michael I.; Dhanda, Anu; Tian, Ya-Min; Masson, Norma; Hamilton, Donald L.; Jaakkola, Panu; Barstead, Robert; Hodgkin, Jonathan; Maxwell, Patrick H.; Pugh, Christopher W.; Schofield, Christopher J.; Ratcliffe, Peter J.

CORPORATE SOURCE: The Henry Wellcome Building of Genomic Medicine, Oxford, OX3 7BN, UK

SOURCE: Cell (Cambridge, MA, United States) (2001), 107(1), 43-54

CODEN: CELLB5; ISSN: 0092-8674

PUBLISHER: Cell Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB HIF is a transcriptional complex that plays a central role in mammalian oxygen homeostasis. Recent studies have defined posttranslational modification by prolyl hydroxylation as a key regulatory event that targets HIF- $\alpha$  subunits for proteasomal destruction via the von Hippel-Lindau ubiquitylation complex. Here, we define a conserved HIF-VHL- \*\*\*prolyl\*\*\* \*\*\*hydroxylase\*\*\* pathway in \*\*\*C\*\*\*. \*\*\*elegans\*\*\*, and use a genetic approach to identify EGL-9 as a dioxygenase that regulates HIF by prolyl hydroxylation. In mammalian cells, we show that the HIF-prolyl hydroxylases are represented by a series of isoforms bearing a conserved 2-histidine-1-carboxylate iron coordination motif at the catalytic site. Direct modulation of recombinant enzyme activity by graded hypoxia, iron chelation, and cobaltous ions mirrors the characteristics of HIF induction in vivo, fulfilling requirements for these enzymes being oxygen sensors that regulate HIF.

REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

ACCESSION NUMBER: 2000:324546 CAPLUS  
 DOCUMENT NUMBER: 133:86929  
 TITLE: Prolyl 4-hydroxylase is an essential procollagen-modifying enzyme required for exoskeleton formation and the maintenance of body shape in the nematode *Caenorhabditis elegans*  
 AUTHOR(S): Winter, Alan D.; Page, Antony P.  
 CORPORATE SOURCE: Wellcome Centre for Molecular Parasitology, Anderson College, The University of Glasgow, Glasgow, G11 6NU, UK  
 SOURCE: Molecular and Cellular Biology (2000), 20(11), 4084-4093  
 CODEN: MCEBD4; ISSN: 0270-7306  
 PUBLISHER: American Society for Microbiology  
 DOCUMENT TYPE: Journal  
 LANGUAGE: English

AB The multienzyme complex prolyl 4-hydroxylase catalyzes the hydroxylation of proline residues and acts as a chaperone during collagen synthesis in multicellular organisms. The .beta. subunit of this complex is identical to protein disulfide isomerase (PDI). The free-living nematode *C. elegans* is encased in a collagenous exoskeleton and represents an excellent model for the study of collagen biosynthesis and extracellular matrix formation. In this study, we examd. prolyl 4-hydroxylase .alpha.-subunit (PHY; EC 1.14.11.2)- and .beta.-subunit (PDI; EC 5.3.4.1)-encoding genes with respect to their role in collagen modification and formation of the *C. elegans* exoskeleton. We identified genes encoding 2 PHYs and a single assocd. PDI and showed that all 3 are expressed in collagen-synthesizing ectodermal cells at times of maximal collagen synthesis. Disruption of the pdi gene via RNA interference resulted in embryonic lethality. Similarly, the combined phy genes are required for embryonic development. Interference with phy-1 resulted in a morphol. dumpy phenotype, which we detd. to be identical to the uncharacterized dpy-18 locus. Two dpy-18 mutant strains were shown to have null alleles for phy-1 and to have a reduced hydroxyproline content in their exoskeleton collagens. This study demonstrates in vivo that this enzyme complex plays a central role in extracellular matrix formation and is essential for normal metazoan development.

REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

ACCESSION NUMBER: 79021663 MEDLINE  
 DOCUMENT NUMBER: 79021663 PubMed ID: 212107  
 TITLE: In vitro translation of nematode cuticular collagens.  
 AUTHOR: Noble S; Leushner J; Pasternak J  
 SOURCE: BIOCHIMICA ET BIOPHYSICA ACTA, (1978 Aug 23) 520 (1) 219-28.  
 Journal code: 0217513. ISSN: 0006-3002.  
 PUB. COUNTRY: Netherlands  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 197812  
 ENTRY DATE: Entered STN: 19900314  
 Last Updated on STN: 19900314  
 Entered Medline: 19781220

AB Phenanthroline treatment of growing cultures of the free-living \*\*\*nematode\*\*\* *Panagrellus silusiae* was used to lower the degree of hydroxylation of nascent collagen chains at the polysomal level. Under these conditions, the bound pentasome-hexasome fraction provided substrate for \*\*\*prolyl\*\*\* \*\*\*hydroxylase\*\*\*. When this polysomal fraction was subsequently tested in a cell-free wheat germ system, collagenase-susceptible translation products were observed after sodium dodecyl sulfate-acrylamide gel electrophoresis. The electrophoretic mobilities of each of these four major collagen products were similar to four collagens that are isolated from intact cuticles. In addition, purified polysomal RNA that adhered to unmodified cellulose directed the synthesis of four pepsin-resistant polypeptides that had molecular weights that coincided with four pepsin-resistant collagens that can be purified from the cuticle of this species. Thus, the polysomal site of the messenger RNAs for the cuticular collagens of *P. silusiae* was located. Although precursor forms of the cuticular collagens were not produced in the cell-free system, the question whether additional amino acid segments occur on the primary translational products of the cuticular collagens in vivo remains open.

L10 ANSWER 4 OF 6 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 3  
 ACCESSION NUMBER: 1978:185080 CAPLUS  
 DOCUMENT NUMBER: 88:185080  
 TITLE: Partial purification and characterization of  
           \*\*\*prolyl\*\*\*        \*\*\*hydroxylase\*\*\* from the  
           free-living        \*\*\*nematode\*\*\* Panagrellus silusiae  
 AUTHOR(S): Leushner, J. R. A.; Pasternak, J.  
 CORPORATE SOURCE: Dep. Biol., Univ. Waterloo, Waterloo, Ont., Can.  
 SOURCE: Can. J. Zool. (1978), 56(2), 159-65  
           CODEN: CJZOAG; ISSN: 0008-4301  
 DOCUMENT TYPE: Journal  
 LANGUAGE: English  
 AB        \*\*\*prolyl\*\*\*        \*\*\*hydroxylase\*\*\* (I) was partially purified from the  
           \*\*\*nematode\*\*\* P. silusiae and its physicochem. and biol. properties  
 were studied. I purifn. involved (NH<sub>4</sub>)<sub>2</sub>SO<sub>4</sub> pptn. and Ca phosphate gel ion  
 exchange from Triton X-100-treated Panagrellus homogenates. Gel  
 filtration indicated a mol. wt. of .apprx.285,000; acrylamide  
 electrophoresis showed the component to be comprised of subunits having  
 mol. wts. of .apprx.67,000. I activity was dependent on  
 .alpha.-ketoglutarate, Fe<sup>2+</sup>, ascorbate, catalase, O<sub>2</sub>, and dithiothreitol.  
 Activity was inhibited by .alpha.,.alpha.-dipyridyl, phenanthroline, and  
 polyproline. The Km value for the substrate was 80 .mu.g.

L10 ANSWER 5 OF 6 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
 ACCESSION NUMBER: 1978:67206 BIOSIS  
 DOCUMENT NUMBER: BR15:10706  
 TITLE: PROTO COLLAGEN        \*\*\*PROLYL\*\*\*        \*\*\*HYDROXYLASE\*\*\* IN  
           THE FREE LIVING        \*\*\*NEMATODE\*\*\* PANAGRELLUS-SILUSIAE.  
 AUTHOR(S): LEUSHNER J R A; PASTERNAK J J  
 SOURCE: Proc. Can. Fed. Biol. Soc., (1976) 19, 98.  
           CODEN: PCBSA2.  
 DOCUMENT TYPE: Conference  
 FILE SEGMENT: BR; OLD  
 LANGUAGE: Unavailable

L10 ANSWER 6 OF 6 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.  
 ACCESSION NUMBER: 76171264 EMBASE  
 DOCUMENT NUMBER: 1976171264  
 TITLE: Programmed synthesis of collagen during postembryonic  
           development of the nematode Panagrellus silusiae.  
 AUTHOR: Leushner J.; Pasternak J.  
 CORPORATE SOURCE: Dept. Biol., Univ. Waterloo, Canada  
 SOURCE: Developmental Biology, (1975) 47/1 (68-80).  
           CODEN: DEBIAO  
 DOCUMENT TYPE: Journal  
 FILE SEGMENT: 029 Clinical Biochemistry  
                   021 Developmental Biology and Teratology  
 LANGUAGE: English  
 AB        The relative rate of collagen synthesis in the free living  
           \*\*\*nematode\*\*\* Panagrellus silusiae during postembryonic development was  
 found to be discontinuous by measuring either the incorporation of tritium  
 into material extracted as collagen or the amount of collagen bound  
 tritiated proline and hydroxyproline after 2 hr incubations of whole worms  
 with [3H]proline. A peak of collagen production preceded each of the three  
 molts that were examined. Moreover, protocollagen        \*\*\*prolyl\*\*\*  
           \*\*\*hydroxylase\*\*\* activity during each intermolt period paralleled the  
 pattern of collagen synthesis. On the other hand, a triphasic pattern was  
 not observed when noncollagenous proteins were labeled with either  
 [3H]tryptophan or [3H]leucine. In addition, the level of soluble  
 radioactive proline that accumulates in whole organisms after 2 hr  
 incubation periods did not fluctuate appreciably during postembryonic  
 development. The mean ratio of hydroxyproline to proline in a number of  
 collagen samples extracted at various times during the maturation phase  
 was 0.113 .+- 0.040. Pulse and chase experiments with [3H]proline  
 indicated that most of the collagen synthesized during a peak period is  
 lost after the second ecdysis following the labeling interval. In  
 contrast, a considerable proportion of the collagen synthesized during  
 nonpeak periods is retained throughout the postembryonic period. It is  
 postulated that the modulated pattern of collagen biosynthesis in  
 Panagrellus reflects, for the most part, a quantitative regulation of the  
 production of cuticular collagen during postembryonic development.

=> D HIS

(FILE 'HOME' ENTERED AT 14:44:43 ON 18 JUL 2002)

FILE 'MEDLINE' ENTERED AT 14:44:53 ON 18 JUL 2002  
L1 19214 S NEMATODE OR C.ELEGANS OR ?ELEGANS  
L2 509 S PROLYL ?HYDROXYLASE  
L3 2 S L1 (S) L2  
L4 2 S L1 (L) L2  
L5 7 S DPY-18  
L6 103 S DPY-18 OR DPY  
L7 59 S L1 (L) L6  
L8 0 S L7 AND L2

FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE' ENTERED AT 14:49:21 ON 18 JUL 2002  
L9 11 S L3  
L10 6 DUP REM L9 (5 DUPLICATES REMOVED)

=> S L5  
L11 25 L5

=> S EMBRYONAL LETHAL PHENOTYPE  
L12 0 EMBRYONAL LETHAL PHENOTYPE

=> S EMBRYONIC LETHAL PHENOTYPE  
L13 283 EMBRYONIC LETHAL PHENOTYPE

=> S EMBRYON? LETHAL PHENOTYPE  
L14 290 EMBRYON? LETHAL PHENOTYPE

=> S EMBRYON? LETHA? PHENOTYPE  
L15 290 EMBRYON? LETHA? PHENOTYPE

=> S L1 (s) L15  
L16 35 L1 (S) L15

=> S L16 AND L2  
L17 0 L16 AND L2

=> S L1 (s) L15 (s) L2  
L18 0 L1 (S) L15 (S) L2

=> S L1 (L) L15 (L) L2  
L19 0 L1 (L) L15 (L) L2

=> D HIS

(FILE 'HOME' ENTERED AT 14:44:43 ON 18 JUL 2002)

FILE 'MEDLINE' ENTERED AT 14:44:53 ON 18 JUL 2002  
L1 19214 S NEMATODE OR C.ELEGANS OR ?ELEGANS  
L2 509 S PROLYL ?HYDROXYLASE  
L3 2 S L1 (S) L2  
L4 2 S L1 (L) L2  
L5 7 S DPY-18  
L6 103 S DPY-18 OR DPY  
L7 59 S L1 (L) L6  
L8 0 S L7 AND L2

FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE' ENTERED AT 14:49:21 ON 18 JUL 2002  
L9 11 S L3  
L10 6 DUP REM L9 (5 DUPLICATES REMOVED)  
L11 25 S L5  
L12 0 S EMBRYONAL LETHAL PHENOTYPE  
L13 283 S EMBRYONIC LETHAL PHENOTYPE  
L14 290 S EMBRYON? LETHAL PHENOTYPE  
L15 290 S EMBRYON? LETHA? PHENOTYPE  
L16 35 S L1 (S) L15  
L17 0 S L16 AND L2  
L18 0 S L1 (S) L15 (S) L2  
L19 0 S L1 (L) L15 (L) L2

=> DUP REM L16  
PROCESSING COMPLETED FOR L16  
L20 12 DUP REM L16 (23 DUPLICATES REMOVED)

=> S L6  
L21 421 L6

=> S L21 (s) L1

=> S L21 AND L22  
L23 222 L21 AND L22

=> S L1 (S) L15 (S) L21  
L24 0 L1 (S) L15 (S) L21

=> S L1 (L) L15 (L) L21  
L25 0 L1 (L) L15 (L) L21

=> S L1 AND L15 AND L21  
L26 1 L1 AND L15 AND L21

=> D

L26 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS  
AN 1998:454514 CAPLUS  
DN 129:212270  
TI Isolation and characterization of lethal mutation near the unc-29 (LG I)  
region of Caenorhabditis \*\*\*elegans\*\*\*  
AU Lee, Jinsook; Ahnn, Joohong  
CS Department of Life Science, Kwangju Institute of Science and Technology,  
Kwangju, 506-712, S. Korea  
SO Korean Journal of Biological Sciences (1998), 2(1), 123-131  
CODEN: KJBSFZ; ISSN: 1226-5071  
PB Korean Association of Biological Sciences  
DT Journal  
LA English

=> D IBIB ABS

L26 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS  
ACCESSION NUMBER: 1998:454514 CAPLUS  
DOCUMENT NUMBER: 129:212270  
TITLE: Isolation and characterization of lethal mutation near  
the unc-29 (LG I) region of Caenorhabditis  
\*\*\*elegans\*\*\*  
AUTHOR(S): Lee, Jinsook; Ahnn, Joohong  
CORPORATE SOURCE: Department of Life Science, Kwangju Institute of  
Science and Technology, Kwangju, 506-712, S. Korea  
SOURCE: Korean Journal of Biological Sciences (1998), 2(1),  
123-131  
CODEN: KJBSFZ; ISSN: 1226-5071  
PUBLISHER: Korean Association of Biological Sciences  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB The unc-29 region on the chromosome I of Caenorhabditis \*\*\*elegans\*\*\*  
has been mutagenized in order to obtain lethal mutations. In this screen,  
the uncoordinated phenotype of unc-29 (e193) mutant was used to identify  
any lethal mutations closely linked to the unc-29 gene, which encodes a  
subunit of nicotinic acetylcholine receptors. The authors have isolated  
six independent mutations (jh1 to jh6) out of approx. 5,200 Et  
methanesulfonate (EMS) treated haploids. Four of the six mutations  
demonstrated \*\*\*embryonic\*\*\* \*\*\*lethal\*\*\* \*\*\*phenotypes\*\*\*,  
while the other two showed embryonic and larval lethal phenotypes.  
Terminal phenotypes obsd. in two mutations (jh1 and jh2) indicated  
developmental defects specific to posterior part of embryos which appeared  
similar to the phenotypes obsd. in nob (no back end) mutants. Another  
mutation (jh4) resulted in an interesting phenotype of body-wall muscle  
degeneration at larval stage. These mutations were mapped by using  
three-factor crosses and deficiency mutants in this region. Here the  
authors report genetic anal. and characterization of these lethal  
mutations.

=> D HIS

(FILE 'HOME' ENTERED AT 14:44:43 ON 18 JUL 2002)

FILE 'MEDLINE' ENTERED AT 14:44:53 ON 18 JUL 2002

L1 19214 S NEMATODE OR C.ELEGANS OR ?ELEGANS  
L2 509 S PROLYL ?HYDROXYLASE  
L3 2 S L1 (S) L2  
L4 2 S L1 (L) L2  
L5 7 S DPY-18

L7 59 S L1 (L) L6  
L8 0 S L7 AND L2

FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE' ENTERED AT 14:49:21 ON 18 JUL 2002

L9 11 S L3  
L10 6 DUP REM L9 (5 DUPLICATES REMOVED)  
L11 25 S L5  
L12 0 S EMBRYONAL LETHAL PHENOTYPE  
L13 283 S EMBRYONIC LETHAL PHENOTYPE  
L14 290 S EMBRYON? LETHAL PHENOTYPE  
L15 290 S EMBRYON? LETHA? PHENOTYPE  
L16 35 S L1 (S) L15  
L17 0 S L16 AND L2  
L18 0 S L1 (S) L15 (S) L2  
L19 0 S L1 (L) L15 (L) L2  
L20 12 DUP REM L16 (23 DUPLICATES REMOVED)  
L21 421 S L6  
L22 222 S L21 (S) L1  
L23 222 S L21 AND L22  
L24 0 S L1 (S) L15 (S) L21  
L25 0 S L1 (L) L15 (L) L21  
L26 1 S L1 AND L15 AND L21

=> DUP REM L22  
PROCESSING COMPLETED FOR L22  
L27 99 DUP REM L22 (123 DUPLICATES REMOVED)

=> S L1 AND L11  
L28 21 L1 AND L11

=> S L27 AND L28  
L29 8 L27 AND L28

=> DUP REM L29  
PROCESSING COMPLETED FOR L29  
L30 8 DUP REM L29 (0 DUPLICATES REMOVED)

=> D 1-8 IBIB ABS

L30 ANSWER 1 OF 8 MEDLINE  
ACCESSION NUMBER: 2002354365 IN-PROCESS  
DOCUMENT NUMBER: 22092148 Pubmed ID: 12097347  
TITLE: High-Throughput Gene Mapping in Caenorhabditis  
\*\*\*elegans\*\*\*  
AUTHOR: Swan Kathryn A; Curtis Damian E; McKusick Kathleen B;  
Voinov Alexander V; Mapa Felipa A; Cancilla Michael R  
CORPORATE SOURCE: Exelixis, Inc., South San Francisco, California 94083-0511,  
USA.  
SOURCE: GENOME RESEARCH, (2002 Jul) 12 (7) 1100-5.  
Journal code: 9518021. ISSN: 1088-9051.  
PUB. COUNTRY: United States  
Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals  
ENTRY DATE: Entered STN: 20020707  
Last Updated on STN: 20020707

AB Positional cloning of mutations in model genetic systems is a powerful method for the identification of targets of medical and agricultural importance. To facilitate the high-throughput mapping of mutations in Caenorhabditis \*\*\*elegans\*\*\*, we have identified a further 9602 putative new single nucleotide polymorphisms (SNPs) between two \*\*\*C\*\*\* strains, Bristol N2 and the Hawaiian mapping strain CB4856, by sequencing inserts from a CB4856 genomic DNA library and using an informatics pipeline to compare sequences with the canonical N2 genomic sequence. When combined with data from other laboratories, our marker set of 17,189 SNPs provides even coverage of the complete worm genome. To date, we have confirmed >1099 evenly spaced SNPs (one every 91 +/- 56 kb) across the six chromosomes and validated the utility of our SNP marker set and new fluorescence polarization-based genotyping methods for systematic and high-throughput identification of genes in \*\*\*C\*\*\*. \*\*\*elegans\*\*\* by cloning several proprietary genes. We illustrate our approach by recombination mapping and confirmation of the mutation in the cloned gene, \*\*\*dpy\*\*\* - \*\*\*18\*\*\*. [The sequence data described in this paper have been submitted to the NCBI dbSNP data library under accession nos. 4388625-4389689 and GenBank dbSTS under accession nos. 973810-974874. The following individuals and institutions kindly provided

L30 ANSWER 2 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
ACCESSION NUMBER: 2002:141383 BIOSIS  
DOCUMENT NUMBER: PREV200200141383  
TITLE: The T-box factor MLS-1 acts as a molecular switch during  
specification of nonstriated muscle in \*\*\*C\*\*\* .  
\*\*\*elegans\*\*\*  
AUTHOR(S): Kostas, Stephen A.; Fire, Andrew (1)  
CORPORATE SOURCE: (1) Department of Embryology, Carnegie Institution of  
Washington, Baltimore, MD, 21210: fire@ciwemb.edu USA  
SOURCE: Genes & Development, (January 15, 2002) Vol. 16, No. 2, pp.  
257-269. <http://www.genesdev.org/>. print.  
ISSN: 0890-9369.  
DOCUMENT TYPE: Article  
LANGUAGE: English

AB we have isolated mutations in a gene mls-1 that is required for proper  
specification of nonstriated muscle fates in Caenorhabditis  
\*\*\*elegans\*\*\*. Loss of MLS-1 activity causes uterine muscle precursors  
to forego their normal fates, instead differentiating as vulval muscles.  
We have cloned mls-1 and shown that the product is a member of the T-box  
family of transcriptional regulators. MLS-1 acts as a cell fate  
determinant in that ectopic expression can transform other cell types to  
uterine muscle precursors. Uterine muscle patterning is executed by  
regulation of MLS-1 at several different levels. The mls-1 promoter is  
activated by the \*\*\*C\*\*\* . \*\*\*elegans\*\*\* orthologs of Twist and  
Daughterless, but is only active in a subset of the lineage where these  
two transcription factors are present. mls-1 activity also appears to be  
regulated by posttranscriptional processes, as expression occurs in both  
uterine and vulval muscle precursors.

L30 ANSWER 3 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
ACCESSION NUMBER: 2002:68425 BIOSIS  
DOCUMENT NUMBER: PREV200200068425  
TITLE: Role of \*\*\*C\*\*\* . \*\*\*elegans\*\*\* lin-40 MTA in vulval  
fate specification and morphogenesis.  
AUTHOR(S): Chen, Zhe; Han, Min (1)  
CORPORATE SOURCE: (1) Department of Molecular, Cellular and Developmental  
Biology, Howard Hughes Medical Institute, University of  
Colorado at Boulder, Boulder, CO, 80309: mhan@colorado.edu  
USA  
SOURCE: Development (Cambridge), (December, 2001) Vol. 128, No. 23,  
pp. 4911-4921. <http://dev.biologists.org/current.shtml>.  
print.  
ISSN: 0950-1991.  
DOCUMENT TYPE: Article  
LANGUAGE: English

AB Vulval differentiation in Caenorhabditis \*\*\*elegans\*\*\* involves  
several fundamental cellular events, including cell fusion, division and  
migration. We have characterized the role of the lin-40 (also known as  
egr-1) gene in these cellular processes. LIN-40 is homologous to the  
metastasis-associated factor 1 (MTA1) in mammals, which has been  
identified as a component of the nucleosome remodeling and histone  
deacetylation (NuRD) complex that functions as a transcriptional  
co-repressor. We show here that lin-40 negatively regulates vulval fate  
specification at least partly by promoting cell fusion between the vulval  
precursor cells and the hypodermal syncytium at an early larval stage.  
This inhibitory function of lin-40 might be carried out by downregulating  
lin-39 Hox expression. We also show that lin-40 is specifically required  
for cell divisions along the transverse orientation during vulval  
morphogenesis.

L30 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2002 ACS  
ACCESSION NUMBER: 2000:296070 CAPLUS  
DOCUMENT NUMBER: 133:233493  
TITLE: Prolyl 4-hydroxylase is required for viability and  
morphogenesis in Caenorhabditis \*\*\*elegans\*\*\*  
AUTHOR(S): Friedman, Lisa; Higgin, Joshua J.; Moulder, Gary;  
Barstead, Robert; Raines, Ronald T.; Kimble, Judith  
CORPORATE SOURCE: Department of Biochemistry, University of Wisconsin,  
Madison, WI, 53706, USA  
SOURCE: Proceedings of the National Academy of Sciences of the  
United States of America (2000), 97(9), 4736-4741  
CODEN: PNASA6; ISSN: 0027-8424  
PUBLISHER: National Academy of Sciences

LANGUAGE: English

AB The genome of *Caenorhabditis elegans* possesses two genes, *dpy-18* and *phy-2*, that encode .alpha. subunits of the enzyme prolyl 4-hydroxylase. The authors have generated deletions within each gene to eliminate prolyl 4-hydroxylase activity from the animal. The *dpy-18* mutant has an aberrant body morphol., consistent with a role of prolyl 4-hydroxylase in formation of the body cuticle. The *phy-2* mutant is phenotypically wild type. However, the *dpy-18*; *phy-2* double mutant is not viable, suggesting an essential role for prolyl 4-hydroxylase that is normally accomplished by either *dpy-18* or *phy-2*. The effects of the double mutation were mimicked by small-mol. inhibitors of prolyl 4-hydroxylase, validating the genetic results and suggesting that *C. elegans* can serve as a model system for the discovery of new inhibitors.

REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L30 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2000:324546 CAPLUS

DOCUMENT NUMBER: 133:86929

TITLE: Prolyl 4-hydroxylase is an essential procollagen-modifying enzyme required for exoskeleton formation and the maintenance of body shape in the *Caenorhabditis elegans*

AUTHOR(S): Winter, Alan D.; Page, Antony P.

CORPORATE SOURCE: Wellcome Centre for Molecular Parasitology, Anderson College, The University of Glasgow, Glasgow, G11 6NU, UK

SOURCE: Molecular and Cellular Biology (2000), 20(11), 4084-4093

PUBLISHER: American Society for Microbiology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The multienzyme complex prolyl 4-hydroxylase catalyzes the hydroxylation of proline residues and acts as a chaperone during collagen synthesis in multicellular organisms. The .beta. subunit of this complex is identical to protein disulfide isomerase (PDI). The free-living *C. elegans* is encased in a collagenous exoskeleton and represents an excellent model for the study of collagen biosynthesis and extracellular matrix formation. In this study, we examd. prolyl 4-hydroxylase .alpha.-subunit (PHY; EC 1.14.11.2)- and .beta.-subunit (PDI; EC 5.3.4.1)-encoding genes with respect to their role in collagen modification and formation of the *C. elegans* exoskeleton. We identified genes encoding 2 PHYs and a single assocd. PDI and showed that all 3 are expressed in collagen-synthesizing ectodermal cells at times of maximal collagen synthesis. Disruption of the *pdi* gene via RNA interference resulted in embryonic lethality. Similarly, the combined *phy* genes are required for embryonic development. Interference with *phy-1* resulted in a morphol. dumphy phenotype, which we detd. to be identical to the uncharacterized *dpy-18* locus. Two *dpy-18* mutant strains were shown to have null alleles for *phy-1* and to have a reduced hydroxyproline content in their exoskeleton collagens. This study demonstrates in vivo that this enzyme complex plays a central role in extracellular matrix formation and is essential for normal metazoan development.

REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L30 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2000:550782 CAPLUS

DOCUMENT NUMBER: 134:247771

TITLE: *dpy-18* encodes an .alpha.-subunit of prolyl-4-hydroxylase in *Caenorhabditis elegans*

AUTHOR(S): Hill, Katherine L.; Harfe, Brian D.; Dobbins, Carey A.; L'Hernault, Steven W.

CORPORATE SOURCE: Program in Genetics and Molecular Biology, Graduate Division of Biological and Biomedical Sciences, Emory University, Atlanta, GA, 30322, USA

SOURCE: Genetics (2000), 155(3), 1139-1148

PUBLISHER: Genetics Society of America

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Collagen is an extracellular matrix (ECM) component encoded by a large



post-translationally modified by prolyl-4-hydroxylase (EC 1.14.11.2) before secretion and participation in ECM formation. Therefore, collagen processing and regulation can be studied by examg. this required interaction of prolyl-4-hydroxylase with procollagen. High-resoln. polymorphism mapping was used to place the Caenorhabditis \*\*\*elegans\*\*\* dpy gene on the phys. map, and we show that it encodes a prolyl-4-hydroxylase .alpha. catalytic subunit. The Dpy phenotype of \*\*\*dpy\*\*\* - \*\*\*18\*\*\* (e364) amber mutants is more severe when this mutation is in trans to the noncomplementing deficiency tdf7, while the \*\*\*dpy\*\*\* - \*\*\*18\*\*\* (e499) deletion mutant exhibits the same phenotype as \*\*\*dpy\*\*\* - \*\*\*18\*\*\* (e499)/tdf7. Furthermore, \*\*\*dpy\*\*\* - \*\*\*18\*\*\* RNA interference (RNAi) in wild-type worms results in Dpy progeny, while \*\*\*dpy\*\*\* - \*\*\*18\*\*\* (RNAi) in \*\*\*dpy\*\*\* - \*\*\*18\*\*\* (e499) mutants does not alter the Dpy phenotype of their progeny. These observations suggest that the \*\*\*dpy\*\*\* - \*\*\*18\*\*\* null phenotype is Dpy. A \*\*\*dpy\*\*\* - \*\*\*18\*\*\* ::gfp promoter fusion construct is expressed throughout the hypodermis within the cells that abundantly produce the cuticle collagens, as well as in certain head and posterior neurons. While prolyl-4-hydroxylase has been studied extensively by biochem. techniques, this is the first report of a mutationally defined prolyl-4-hydroxylase in any animal.

REFERENCE COUNT: 59 THERE ARE 59 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L30 ANSWER 7 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
 ACCESSION NUMBER: 2000:323602 BIOSIS  
 DOCUMENT NUMBER: PREV200000323602  
 TITLE: Mutations with sensory ray defect unmask cuticular glycoprotein antigens in Caenorhabditis \*\*\*elegans\*\*\* male tail.  
 AUTHOR(S): Ko, Frankie C. F.; Chow, King L. (1)  
 CORPORATE SOURCE: (1) Department of Biology, Hong Kong University of Science and Technology, Clear Water Bay, Kowloon, Hong Kong China  
 SOURCE: Development Growth & Differentiation, (Feb., 2000) vol. 42, No. 1, pp. 69-77. print.  
 ISSN: 0012-1592.

DOCUMENT TYPE: Article  
 LANGUAGE: English  
 SUMMARY LANGUAGE: English

AB Caenorhabditis \*\*\*elegans\*\*\* male tail has nine pairs of bilaterally symmetric ray processes extended into a cuticular fan. The formation of these structures involves both cell lineage differentiation and cellular morphogenesis. Nine mutations were examined, all of which presented an amorphous ray phenotype. Glycoconjugates carrying an N-acetylglucosamine (GlcNAc) epitope were detected at a high level in their male bursa. It was shown that these antigens are not responsible for the morphological defects. It was further demonstrated that these ram and mab gene products represent critical components for male tail cuticle organization. Mutations of them abolish the integrity of the male bursal cuticle and unmask the underlying GlcNAc epitope.

L30 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2002 ACS  
 ACCESSION NUMBER: 1991:116067 CAPLUS  
 DOCUMENT NUMBER: 114:116067  
 TITLE: Properties of a class of genes required for ray morphogenesis in Caenorhabditis \*\*\*elegans\*\*\*  
 AUTHOR(S): Baird, Scott E.; Emmons, Scott W.  
 CORPORATE SOURCE: Dep. Mol. Genet., Albert Einstein Coll. Med., Bronx, NY, 10461, USA  
 SOURCE: Genetics (1990), 126(2), 335-44  
 CODEN: GENTAE; ISSN: 0016-6731

DOCUMENT TYPE: Journal  
 LANGUAGE: English

AB Eight mutations were identified in Caenorhabditis \*\*\*elegans\*\*\* that define at least 5 terminal differentiation genes (ram genes) whose products are required during the extension of the male-specific ray sensilla. ram Gene mutations result in morphol. abnormalities in the sensory rays but do not appear to interfere with ray functions. A similar ray morphol. phenotype was obsd. in males harboring mutations in 3 previously defined genes, dpy-11, \*\*\*dpy\*\*\* - \*\*\*18\*\*\*, and sqt-1, that also affect body shape. One of these genes, sqt-1, is known to encode a collagen. Mutations in different ram genes failed to complement, suggesting that their gene products functionally interact. For one ram gene, failure to complement was shown to result from haploinsufficiency. Intergenic noncomplementation did not extend to the body morphol. genes. The temp.-sensitive periods of both ram and body morphol. mutations

It is proposed that ram gene products act together in a crit. interaction between the rays and the cuticle required for wild-type ray morphol.

=> D HIS

(FILE 'HOME' ENTERED AT 14:44:43 ON 18 JUL 2002)

FILE 'MEDLINE' ENTERED AT 14:44:53 ON 18 JUL 2002

L1 19214 S NEMATODE OR C.ELEGANS OR ?ELEGANS  
L2 509 S PROLYL ?HYDROXYLASE  
L3 2 S L1 (S) L2  
L4 2 S L1 (L) L2  
L5 7 S DPY-18  
L6 103 S DPY-18 OR DPY  
L7 59 S L1 (L) L6  
L8 0 S L7 AND L2

FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE' ENTERED AT 14:49:21 ON 18 JUL 2002

L9 11 S L3  
L10 6 DUP REM L9 (5 DUPLICATES REMOVED)  
L11 25 S L5  
L12 0 S EMBRYONAL LETHAL PHENOTYPE  
L13 283 S EMBRYONIC LETHAL PHENOTYPE  
L14 290 S EMBRYON? LETHAL PHENOTYPE  
L15 290 S EMBRYON? LETHA? PHENOTYPE  
L16 35 S L1 (S) L15  
L17 0 S L16 AND L2  
L18 0 S L1 (S) L15 (S) L2  
L19 0 S L1 (L) L15 (L) L2  
L20 12 DUP REM L16 (23 DUPLICATES REMOVED)  
L21 421 S L6  
L22 222 S L21 (S) L1  
L23 222 S L21 AND L22  
L24 0 S L1 (S) L15 (S) L21  
L25 0 S L1 (L) L15 (L) L21  
L26 1 S L1 AND L15 AND L21  
L27 99 DUP REM L22 (123 DUPLICATES REMOVED)  
L28 21 S L1 AND L11  
L29 8 S L27 AND L28  
L30 8 DUP REM L29 (0 DUPLICATES REMOVED)

=> LOGOFF HOLD

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

144.04

147.31

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE

TOTAL

ENTRY

SESSION

CA SUBSCRIBER PRICE

-4.96

-4.96

SESSION WILL BE HELD FOR 60 MINUTES

STN INTERNATIONAL SESSION SUSPENDED AT 15:02:46 ON 18 JUL 2002

Connecting via winsock to STN

welcome to STN International! Enter x:X

LOGINID:ssspta1632rrs

PASSWORD:

\* \* \* \* \* RECONNECTED TO STN INTERNATIONAL \* \* \* \* \*

SESSION RESUMED IN FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE'

AT 15:16:44 ON 18 JUL 2002

FILE 'CAPLUS' ENTERED AT 15:16:44 ON 18 JUL 2002

COPYRIGHT (C) 2002 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'MEDLINE' ENTERED AT 15:16:44 ON 18 JUL 2002

FILE 'BIOSIS' ENTERED AT 15:16:44 ON 18 JUL 2002

COPYRIGHT (C) 2002 BIOLOGICAL ABSTRACTS INC.(R)

FILE 'EMBASE' ENTERED AT 15:16:44 ON 18 JUL 2002

COPYRIGHT (C) 2002 Elsevier Science B.V. All rights reserved.

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE

TOTAL

CA SUBSCRIBER PRICE

ENTRY

SION

-4.96

-4.96

=> D HIS

(FILE 'HOME' ENTERED AT 14:44:43 ON 18 JUL 2002)

FILE 'MEDLINE' ENTERED AT 14:44:53 ON 18 JUL 2002

L1 19214 S NEMATODE OR C.ELEGANS OR ?ELEGANS  
L2 509 S PROLYL ?HYDROXYLASE  
L3 2 S L1 (S) L2  
L4 2 S L1 (L) L2  
L5 7 S DPY-18  
L6 103 S DPY-18 OR DPY  
L7 59 S L1 (L) L6  
L8 0 S L7 AND L2

FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE' ENTERED AT 14:49:21 ON 18 JUL 2002

L9 11 S L3  
L10 6 DUP REM L9 (5 DUPLICATES REMOVED)  
L11 25 S L5  
L12 0 S EMBRYONAL LETHAL PHENOTYPE  
L13 283 S EMBRYONIC LETHAL PHENOTYPE  
L14 290 S EMBRYON? LETHAL PHENOTYPE  
L15 290 S EMBRYON? LETHA? PHENOTYPE  
L16 35 S L1 (S) L15  
L17 0 S L16 AND L2  
L18 0 S L1 (S) L15 (S) L2  
L19 0 S L1 (L) L15 (L) L2  
L20 12 DUP REM L16 (23 DUPLICATES REMOVED)  
L21 421 S L6  
L22 222 S L21 (S) L1  
L23 222 S L21 AND L22  
L24 0 S L1 (S) L15 (S) L21  
L25 0 S L1 (L) L15 (L) L21  
L26 1 S L1 AND L15 AND L21  
L27 99 DUP REM L22 (123 DUPLICATES REMOVED)  
L28 21 S L1 AND L11  
L29 8 S L27 AND L28  
L30 8 DUP REM L29 (0 DUPLICATES REMOVED)

=> D HIS

(FILE 'HOME' ENTERED AT 14:44:43 ON 18 JUL 2002)

FILE 'MEDLINE' ENTERED AT 14:44:53 ON 18 JUL 2002

L1 19214 S NEMATODE OR C.ELEGANS OR ?ELEGANS  
L2 509 S PROLYL ?HYDROXYLASE  
L3 2 S L1 (S) L2  
L4 2 S L1 (L) L2  
L5 7 S DPY-18  
L6 103 S DPY-18 OR DPY  
L7 59 S L1 (L) L6  
L8 0 S L7 AND L2

FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE' ENTERED AT 14:49:21 ON 18 JUL 2002

L9 11 S L3  
L10 6 DUP REM L9 (5 DUPLICATES REMOVED)  
L11 25 S L5  
L12 0 S EMBRYONAL LETHAL PHENOTYPE  
L13 283 S EMBRYONIC LETHAL PHENOTYPE  
L14 290 S EMBRYON? LETHAL PHENOTYPE  
L15 290 S EMBRYON? LETHA? PHENOTYPE  
L16 35 S L1 (S) L15  
L17 0 S L16 AND L2  
L18 0 S L1 (S) L15 (S) L2  
L19 0 S L1 (L) L15 (L) L2  
L20 12 DUP REM L16 (23 DUPLICATES REMOVED)  
L21 421 S L6  
L22 222 S L21 (S) L1  
L23 222 S L21 AND L22  
L24 0 S L1 (S) L15 (S) L21  
L25 0 S L1 (L) L15 (L) L21  
L26 1 S L1 AND L15 AND L21

photomorphogenesis and dwarfism (cpd) mutant. Measurements of endogenous brassinosteroid levels by gas chromatog.-mass spectrometry were consistent with this hypothesis. To examine brassinosteroid-regulated gene expression in dpy, we performed cDNA subtractive hybridization and isolated a novel xyloglucan endotransglycosylase that is regulated by brassinosteroid treatment. The curl-3 (cu-3) mutant (Lycopersicon pimpinellifolium [Jusl.] Mill.) shows extreme dwarfism, altered leaf morphol., de-etiolation, and reduced fertility, all strikingly similar to the Arabidopsis mutant brassinosteroid insensitive 1 (bril). Primary root elongation of wild-type L. pimpinellifolium seedlings was strongly inhibited by brassinosteroid application, while cu-3 mutant roots were able to elongate at the same brassinosteroid concn. Moreover, cu-3 mutants retained sensitivity to indole-3-acetic acid, cytokinins, gibberellin, and abscisic acid while showing hypersensitivity to 2,4-dichlorophenoxyacetic acid in the root elongation assay. The cu-3 root response to hormones, coupled with its bril-like phenotype, suggests that cu-3 may also be brassinosteroid insensitive.

REFERENCE COUNT: 57 THERE ARE 57 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L33 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 3  
ACCESSION NUMBER: 1994:209702 CAPLUS  
DOCUMENT NUMBER: 120:209702  
TITLE: Molecular and genetic analyses of the Caenorhabditis elegans dpy-2 and dpy-10 collagen genes: A variety of molecular alterations affect organismal morphology  
AUTHOR(S): Levy, Adam D.; Yang, Jie; Kramer, James M.  
CORPORATE SOURCE: Med. Sch., Northwestern Univ., Chicago, IL, 60611, USA  
SOURCE: Mol. Biol. Cell (1993), 4(8), 803-17  
CODEN: MBCEEV; ISSN: 1059-1524  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB The authors have identified and cloned the Caenorhabditis elegans dpy-2 and dpy-10 genes and detd. that they encode collagens. Genetic data suggested that these genes are important in morphogenesis and possibly other developmental events. These data include the morphol. phenotypes exhibited by mutants, unusual genetic interactions with the sqt-1 collagen gene, and suppression of mutations in the glp-1 and mup-1 genes. The proximity of the dpy-2 and dpy-10 genes (3.5 kilobase) and the structural similarity of their encoded proteins (41% amino acid identity) indicate that dpy-2 and dpy-10 are the result of a gene duplication event. The genes do not, however, appear to be functionally redundant, because a dpy-10 null mutant is not rescued by the dpy-2 gene. In addn., full complementation between dpy-2 and dpy-10 can be demonstrated with all recessive alleles tested in trans. Sequence anal. of several mutant alleles of each gene was performed to det. the nature of the mol. defects that can cause the morphol. phenotypes. Glycine substitutions within the Gly-X-Y portion of the collagens can result in dumpy (Dpy), dumpy, left roller (DLRol), or temp.-sensitive DLRol phenotypes. Dpy-10(cn64), a dominant temp.-sensitive DLRol allele, creates an Arg-to-Cys substitution in the amino non-Gly-X-Y portion of the protein. Three dpy-10 alleles contain Tc1 insertions in the coding region of the gene. Dpy-10(cg36) (DLRol) creates a nonsense codon near the end of the Gly-X-Y region. The nature of this mutation, combined with genetic data, indicates that DLRol is the null phenotype of dpy-10. The **Dpy phenotype** results from reduced function of the dpy-10 collagen gene. The authors' results indicate that a variety of mol. defects in these collagens can result in severe morphol. changes in C. elegans.

the genetic results and suggesting that **C. elegans** can  
serve as a model system for the discovery of new inhibitors.

REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS  
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L30 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2000:324546 CAPLUS

DOCUMENT NUMBER: 133:86929

TITLE: Prolyl 4-hydroxylase is an essential  
procollagen-modifying enzyme required for exoskeleton  
formation and the maintenance of body shape in the  
**nematode** *Caenorhabditis elegans*

AUTHOR(S): Winter, Alan D.; Page, Antony P.

CORPORATE SOURCE: Wellcome Centre for Molecular Parasitology, Anderson  
College, The University of Glasgow, Glasgow, G11 6NU,  
UK

SOURCE: Molecular and Cellular Biology (2000), 20(11),  
4084-4093

CODEN: MCEBD4; ISSN: 0270-7306

PUBLISHER: American Society for Microbiology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The multienzyme complex prolyl 4-hydroxylase catalyzes the hydroxylation  
of proline residues and acts as a chaperone during collagen synthesis in  
multicellular organisms. The .beta. subunit of this complex is identical  
to protein disulfide isomerase (PDI). The free-living **nematode**  
**C. elegans** is encased in a collagenous exoskeleton and  
represents an excellent model for the study of collagen biosynthesis and  
extracellular matrix formation. In this study, we examd. prolyl  
4-hydroxylase .alpha.-subunit (PHY; EC 1.14.11.2)- and .beta.-subunit  
(PDI; EC 5.3.4.1)-encoding genes with respect to their role in collagen  
modification and formation of the **C. elegans**  
exoskeleton. We identified genes encoding 2 PHYs and a single assocd. PDI  
and showed that all 3 are expressed in collagen-synthesizing ectodermal  
cells at times of maximal collagen synthesis. Disruption of the pdi gene  
via RNA interference resulted in embryonic lethality. Similarly, the  
combined phy genes are required for embryonic development. Interference  
with phy-1 resulted in a morphol. dumpy phenotype, which we detd. to be  
identical to the uncharacterized **dpy-18** locus. Two  
**dpy-18** mutant strains were shown to have null alleles  
for phy-1 and to have a reduced hydroxyproline content in their  
exoskeleton collagens. This study demonstrates in vivo that this enzyme  
complex plays a central role in extracellular matrix formation and is  
essential for normal metazoan development.

REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS  
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L30 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2000:550782 CAPLUS

DOCUMENT NUMBER: 134:247771

TITLE: **dpy-18** encodes an .alpha.-subunit  
of prolyl-4-hydroxylase in *Caenorhabditis*  
**elegans**

AUTHOR(S): Hill, Katherine L.; Harfe, Brian D.; Dobbins, Carey  
A.; L'Hernault, Steven W.

CORPORATE SOURCE: Program in Genetics and Molecular Biology, Graduate  
Division of Biological and Biomedical Sciences, Emory  
University, Atlanta, GA, 30322, USA

SOURCE: Genetics (2000), 155(3), 1139-1148

CODEN: GENTAE; ISSN: 0016-6731

PUBLISHER: Genetics Society of America

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Collagen is an extracellular matrix (ECM) component encoded by a large

multigene family in multicellular animals. Procollagen is post-translationally modified by prolyl-4-hydroxylase (EC 1.14.11.2) before secretion and participation in ECM formation. Therefore, collagen processing and regulation can be studied by examg. this required interaction of prolyl-4-hydroxylase with procollagen. High-resoln. polymorphism mapping was used to place the *Caenorhabditis elegans* **dpy-18** gene on the phys. map, and we show that it encodes a prolyl-4-hydroxylase .alpha. catalytic subunit. The Dpy phenotype of **dpy-18**(e364) amber mutants is more severe when this mutation is in trans to the noncomplementing deficiency tDf7, while the **dpy-18**(e499) deletion mutant exhibits the same phenotype as **dpy-18**(e499)/tDf7. Furthermore, **dpy-18** RNA interference (RNAi) in wild-type worms results in Dpy progeny, while **dpy-18** (RNAi) in **dpy-18**(e499) mutants does not alter the Dpy phenotype of their progeny. These observations suggest that the **dpy-18** null phenotype is Dpy. A **dpy-18::gfp** promoter fusion construct is expressed throughout the hypodermis within the cells that abundantly produce the cuticle collagens, as well as in certain head and posterior neurons. While prolyl-4-hydroxylase has been studied extensively by biochem. techniques, this is the first report of a mutationally defined prolyl-4-hydroxylase in any animal.

REFERENCE COUNT: 59 THERE ARE 59 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L30 ANSWER 7 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
 ACCESSION NUMBER: 2000:323602 BIOSIS  
 DOCUMENT NUMBER: PREV200000323602  
 TITLE: Mutations with sensory ray defect unmask cuticular glycoprotein antigens in *Caenorhabditis elegans* male tail.  
 AUTHOR(S): Ko, Frankie C. F.; Chow, King L. (1)  
 CORPORATE SOURCE: (1) Department of Biology, Hong Kong University of Science and Technology, Clear Water Bay, Kowloon, Hong Kong China  
 SOURCE: Development Growth & Differentiation, (Feb., 2000) Vol. 42, No. 1, pp. 69-77. print.  
 ISSN: 0012-1592.

DOCUMENT TYPE: Article  
 LANGUAGE: English  
 SUMMARY LANGUAGE: English

AB *Caenorhabditis elegans* male tail has nine pairs of bilaterally symmetric ray processes extended into a cuticular fan. The formation of these structures involves both cell lineage differentiation and cellular morphogenesis. Nine mutations were examined, all of which presented an amorphous ray phenotype. Glycoconjugates carrying an N-acetylglucosamine (GlcNAc) epitope were detected at a high level in their male bursa. It was shown that these antigens are not responsible for the morphological defects. It was further demonstrated that these ram and mab gene products represent critical components for male tail cuticle organization. Mutations of them abolish the integrity of the male bursal cuticle and unmask the underlying GlcNAc epitope.

L30 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2002 ACS  
 ACCESSION NUMBER: 1991:116067 CAPLUS  
 DOCUMENT NUMBER: 114:116067  
 TITLE: Properties of a class of genes required for ray morphogenesis in *Caenorhabditis elegans*  
 AUTHOR(S): Baird, Scott E.; Emmons, Scott W.  
 CORPORATE SOURCE: Dep. Mol. Genet., Albert Einstein Coll. Med., Bronx, NY, 10461, USA  
 SOURCE: Genetics (1990), 126(2), 335-44  
 CODEN: GENTAE; ISSN: 0016-6731  
 DOCUMENT TYPE: Journal  
 LANGUAGE: English

AB Eight mutations were identified in *Caenorhabditis elegans* that define at least 5 terminal differentiation genes (ram genes) whose products are required during the extension of the male-specific ray sensilla. ram Gene mutations result in morphol. abnormalities in the sensory rays but do not appear to interfere with ray functions. A similar ray morphol. phenotype was obsd. in males harboring mutations in 3 previously defined genes, dpy-11, **dpy-18**, and sqt-1, that also affect body shape. One of these genes, sqt-1, is known to encode a collagen. Mutations in different ram genes failed to complement, suggesting that their gene products functionally interact. For one ram gene, failure to complement was shown to result from haploinsufficiency. Intergenic noncomplementation did not extend to the body morphol. genes. The temp.-sensitive periods of both ram and body morphol. mutations corresponded to the period of development in which ray extension occurs. It is proposed that ram gene products act together in a crit. interaction between the rays and the cuticle required for wild-type ray morphol.

=> D HIS

(FILE 'HOME' ENTERED AT 14:44:43 ON 18 JUL 2002)

FILE 'MEDLINE' ENTERED AT 14:44:53 ON 18 JUL 2002

```
L1      19214 S NEMATODE OR C.ELEGANS OR ?ELEGANS
L2      509 S PROLYL ?HYDROXYLASE
L3      2 S L1 (S) L2
L4      2 S L1 (L) L2
L5      7 S DPY-18
L6      103 S DPY-18 OR DPY
L7      59 S L1 (L) L6
L8      0 S L7 AND L2
```

FILE 'CAPLUS, MEDLINE, BIOSIS, EMBASE' ENTERED AT 14:49:21 ON 18 JUL 2002

```
L9      11 S L3
L10     6 DUP REM L9 (5 DUPLICATES REMOVED)
L11     25 S L5
L12     0 S EMBRYONAL LETHAL PHENOTYPE
L13     283 S EMBRYONIC LETHAL PHENOTYPE
L14     290 S EMBRYON? LETHAL PHENOTYPE
L15     290 S EMBRYON? LETHA? PHENOTYPE
L16     35 S L1 (S) L15
L17     0 S L16 AND L2
L18     0 S L1 (S) L15 (S) L2
L19     0 S L1 (L) L15 (L) L2
L20     12 DUP REM L16 (23 DUPLICATES REMOVED)
L21     421 S L6
L22     222 S L21 (S) L1
L23     222 S L21 AND L22
L24     0 S L1 (S) L15 (S) L21
L25     0 S L1 (L) L15 (L) L21
L26     1 S L1 AND L15 AND L21
L27     99 DUP REM L22 (123 DUPLICATES REMOVED)
L28     21 S L1 AND L11
L29     8 S L27 AND L28
L30     8 DUP REM L29 (0 DUPLICATES REMOVED)
```

=> LOGOFF HOLD

L26 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1998:454514 CAPLUS

DOCUMENT NUMBER: 129:212270

TITLE: Isolation and characterization of lethal mutation near the unc-29 (LG I) region of *Caenorhabditis elegans*

AUTHOR(S): Lee, Jinsook; Ahnn, Joohong

CORPORATE SOURCE: Department of Life Science, Kwangju Institute of Science and Technology, Kwangju, 506-712, S. Korea

SOURCE: Korean Journal of Biological Sciences (1998), 2(1), 123-131

CODEN: KJBSFZ; ISSN: 1226-5071

PUBLISHER: Korean Association of Biological Sciences

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The unc-29 region on the chromosome I of *Caenorhabditis elegans* has been mutagenized in order to obtain lethal mutations. In this screen, the uncoordinated phenotype of unc-29 (e193) mutant was used to identify any lethal mutations closely linked to the unc-29 gene, which encodes a subunit of nicotinic acetylcholine receptors. The authors have isolated six independent mutations (jh1 to jh6) out of approx. 5,200 Et methanesulfonate (EMS) treated haploids. Four of the six mutations demonstrated **embryonic lethal phenotypes**, while the other two showed embryonic and larval lethal phenotypes. Terminal phenotypes obsd. in two mutations (jh1 and jh2) indicated developmental defects specific to posterior part of embryos which appeared similar to the phenotypes obsd. in nob (no back end) mutants. Another mutation (jh4) resulted in an interesting phenotype of body-wall muscle degeneration at larval stage. These mutations were mapped by using three-factor crosses and deficiency mutants in this region. Here the authors report genetic anal. and characterization of these lethal mutations.



regulation of MLS-1 at several different levels. The mls-1 promoter is activated by the **C. elegans** orthologs of Twist and Daughterless, but is only active in a subset of the lineage where these two transcription factors are present. mls-1 activity also appears to be regulated by posttranscriptional processes, as expression occurs in both uterine and vulval muscle precursors.

L30 ANSWER 3 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
ACCESSION NUMBER: 2002:68425 BIOSIS  
DOCUMENT NUMBER: PREV200200068425  
TITLE: Role of **C. elegans** lin-40 MTA in vulval fate specification and morphogenesis.  
AUTHOR(S): Chen, Zhe; Han, Min (1)  
CORPORATE SOURCE: (1) Department of Molecular, Cellular and Developmental Biology, Howard Hughes Medical Institute, University of Colorado at Boulder, Boulder, CO, 80309: mhan@colorado.edu USA  
SOURCE: Development (Cambridge), (December, 2001) Vol. 128, No. 23, pp. 4911-4921. <http://dev.biologists.org/current.shtml>. print.  
ISSN: 0950-1991.  
DOCUMENT TYPE: Article  
LANGUAGE: English

AB Vulval differentiation in *Caenorhabditis elegans* involves several fundamental cellular events, including cell fusion, division and migration. We have characterized the role of the lin-40 (also known as egr-1) gene in these cellular processes. LIN-40 is homologous to the metastasis-associated factor 1 (MTA1) in mammals, which has been identified as a component of the nucleosome remodeling and histone deacetylation (NuRD) complex that functions as a transcriptional co-repressor. We show here that lin-40 negatively regulates vulval fate specification at least partly by promoting cell fusion between the vulval precursor cells and the hypodermal syncytium at an early larval stage. This inhibitory function of lin-40 might be carried out by downregulating lin-39 Hox expression. We also show that lin-40 is specifically required for cell divisions along the transverse orientation during vulval morphogenesis.

L30 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2002 ACS  
ACCESSION NUMBER: 2000:296070 CAPLUS  
DOCUMENT NUMBER: 133:233493  
TITLE: Prolyl 4-hydroxylase is required for viability and morphogenesis in *Caenorhabditis elegans*  
AUTHOR(S): Friedman, Lisa; Higgin, Joshua J.; Moulder, Gary; Barstead, Robert; Raines, Ronald T.; Kimble, Judith  
CORPORATE SOURCE: Department of Biochemistry, University of Wisconsin, Madison, WI, 53706, USA  
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (2000), 97(9), 4736-4741  
CODEN: PNASA6; ISSN: 0027-8424  
PUBLISHER: National Academy of Sciences  
DOCUMENT TYPE: Journal  
LANGUAGE: English

AB The genome of *Caenorhabditis elegans* possesses two genes, **dpv-18** and **phy-2**, that encode .alpha. subunits of the enzyme prolyl 4-hydroxylase. Th authors have generated deletions within each gene to eliminate prolyl 4-hydroxylase activity from the animal. The **dpv-18** mutant has an aberrant body morphol., consistent with a role of prolyl 4-hydroxylase in formation of the body cuticle. The **phy-2** mutant is phenotypically wild type. However, the **dpv-18; phy-2** double mutant is not viable, suggesting an essential role for prolyl 4-hydroxylase that is normally accomplished by either **dpv-18** or **phy-2**. The effects of the double mutation were mimicked by small-mol. inhibitors of prolyl 4-hydroxylase, validating

L30 ANSWER 1 OF 8 MEDLINE  
 ACCESSION NUMBER: 2002354365 IN-PROCESS  
 DOCUMENT NUMBER: 22092148 PubMed ID: 12097347  
 TITLE: High-Throughput Gene Mapping in *Caenorhabditis elegans*.  
 AUTHOR: Swan Kathryn A; Curtis Damian E; McKusick Kathleen B; Voinov Alexander V; Mapa Felipa A; Cancilla Michael R  
 CORPORATE SOURCE: Exelixis, Inc., South San Francisco, California 94083-0511, USA.  
 SOURCE: GENOME RESEARCH, (2002 Jul) 12 (7) 1100-5.  
 Journal code: 9518021. ISSN: 1088-9051.  
 PUB. COUNTRY: United States  
 Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals  
 ENTRY DATE: Entered STN: 20020707  
 Last Updated on STN: 20020707

AB Positional cloning of mutations in model genetic systems is a powerful method for the identification of targets of medical and agricultural importance. To facilitate the high-throughput mapping of mutations in *Caenorhabditis elegans*, we have identified a further 9602 putative new single nucleotide polymorphisms (SNPs) between two *C. elegans* strains, Bristol N2 and the Hawaiian mapping strain CB4856, by sequencing inserts from a CB4856 genomic DNA library and using an informatics pipeline to compare sequences with the canonical N2 genomic sequence. When combined with data from other laboratories, our marker set of 17,189 SNPs provides even coverage of the complete worm genome. To date, we have confirmed >1099 evenly spaced SNPs (one every 91 +/- 56 kb) across the six chromosomes and validated the utility of our SNP marker set and new fluorescence polarization-based genotyping methods for systematic and high-throughput identification of genes in *C. elegans* by cloning several proprietary genes. We illustrate our approach by recombination mapping and confirmation of the mutation in the cloned gene, *dpy-18*. [The sequence data described in this paper have been submitted to the NCBI dbSNP data library under accession nos. 4388625-4389689 and GenBank dbSTS under accession nos. 973810-974874. The following individuals and institutions kindly provided reagents, samples, or unpublished information as indicated in the paper: The *C. elegans* Sequencing Consortium and The *Caenorhabditis* Genetics Center.]

L30 ANSWER 2 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
 ACCESSION NUMBER: 2002:141383 BIOSIS  
 DOCUMENT NUMBER: PREV200200141383  
 TITLE: The T-box factor MLS-1 acts as a molecular switch during specification of nonstriated muscle in *C. elegans*.  
 AUTHOR(S): Kostas, Stephen A.; Fire, Andrew (1)  
 CORPORATE SOURCE: (1) Department of Embryology, Carnegie Institution of Washington, Baltimore, MD, 21210: fire@ciwemb.edu USA  
 SOURCE: Genes & Development, (January 15, 2002) Vol. 16, No. 2, pp. 257-269. <http://www.genesdev.org/>. print.  
 ISSN: 0890-9369.  
 DOCUMENT TYPE: Article  
 LANGUAGE: English

AB We have isolated mutations in a gene *mls-1* that is required for proper specification of nonstriated muscle fates in *Caenorhabditis elegans*. Loss of MLS-1 activity causes uterine muscle precursors to forego their normal fates, instead differentiating as vulval muscles. We have cloned *mls-1* and shown that the product is a member of the T-box family of transcriptional regulators. MLS-1 acts as a cell fate determinant in that ectopic expression can transform other cell types to uterine muscle precursors. Uterine muscle patterning is executed by

\$%^STN;HighlightOn= \*\*\*;HighlightOff=\*\*\* ;

Connecting via winsock to STN

Welcome to STN International! Enter x:X

LOGINID:ssspta1632rrs

PASSWORD:

TERMINAL (ENTER 1, 2, 3, OR ?):2

\* \* \* \* \* welcome to STN International \* \* \* \* \*

NEWS 1 Web Page URLs for STN Seminar Schedule - N. America  
NEWS 2 Jan 25 BLAST(R) searching in REGISTRY available in STN on the web  
NEWS 3 Jan 29 FSTA has been reloaded and moves to weekly updates  
NEWS 4 Feb 01 DKILIT now produced by FIZ Karlsruhe and has a new update  
frequency  
NEWS 5 Feb 19 Access via Tymnet and SprintNet Eliminated Effective 3/31/02  
NEWS 6 Mar 08 Gene Names now available in BIOSIS  
NEWS 7 Mar 22 TOXLIT no longer available  
NEWS 8 Mar 22 TRCTHERMO no longer available  
NEWS 9 Mar 28 US Provisional Priorities searched with P in CA/Caplus  
and USPATFULL  
NEWS 10 Mar 28 LIPINSKI/CALC added for property searching in REGISTRY  
NEWS 11 Apr 02 PAPERCHEM no longer available on STN. Use PAPERCHEM2 instead.  
NEWS 12 Apr 08 "Ask CAS" for self-help around the clock  
NEWS 13 Apr 09 BEILSTEIN: Reload and Implementation of a New Subject Area  
NEWS 14 Apr 09 ZDB will be removed from STN  
NEWS 15 Apr 19 US Patent Applications available in IFICDB, IFIPAT, and IFIADB  
NEWS 16 Apr 22 Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS  
NEWS 17 Apr 22 BIOSIS Gene Names now available in TOXCENTER  
NEWS 18 Apr 22 Federal Research in Progress (FEDRIP) now available  
NEWS 19 Jun 03 New e-mail delivery for search results now available  
NEWS 20 Jun 10 MEDLINE Reload  
NEWS 21 Jun 10 PCTFULL has been reloaded  
NEWS 22 Jul 02 FOREGE no longer contains STANDARDS file segment  
  
NEWS EXPRESS February 1 CURRENT WINDOWS VERSION IS V6.0d,  
CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP),  
AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002  
NEWS HOURS STN Operating Hours Plus Help Desk Availability  
NEWS INTER General Internet Information  
NEWS LOGIN Welcome Banner and News Items  
NEWS PHONE Direct Dial and Telecommunication Network Access to STN  
NEWS WWW CAS world wide web Site (general information)

Enter NEWS followed by the item number or name to see news on that specific topic.

All use of STN is subject to the provisions of the STN Customer agreement. Please note that this agreement limits use to scientific research. Use for software development or design or implementation of commercial gateways or other similar uses is prohibited and may result in loss of user privileges and other penalties.

\* \* \* \* \* STN Columbus \* \* \* \* \*

FILE 'HOME' ENTERED AT 14:44:43 ON 18 JUL 2002

=> LE MEDLINE

LE IS NOT A RECOGNIZED COMMAND

The previous command name entered was not recognized by the system.  
For a list of commands available to you in the current file, enter "HELP COMMANDS" at an arrow prompt (=>).

=> FILE MEDLINE

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

0.21

0.21

FILE 'MEDLINE' ENTERED AT 14:44:53 ON 18 JUL 2002

FILE LAST UPDATED: 17 JUL 2002 (20020717/UP). FILE COVERS 1958 TO DATE.

On June 9, 2002, MEDLINE was reloaded. See HELP RLOAD for details.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2002 vocabulary. Enter HELP THESAURUS for details.

THIS FILE CONTAINS CAS REGISTRY NUMBERS FOR EASY AND ACCURATE SUBSTANCE IDENTIFICATION.

```
=> S NEMATODE OR C.ELEGANS OR ?ELEGANS
    11672 NEMATODE
    4439 NEMATODES
    13874 NEMATODE
        (NEMATODE OR NEMATODES)
    728697 C
    7442 ELEGANS
    2724 C.ELEGANS
        (C(W)ELEGANS)
    7443 ?ELEGANS
L1    19214 NEMATODE OR C.ELEGANS OR ?ELEGANS
```

```
=> S PROLYL?HYDROXYLASE
'?' TRUNCATION SYMBOL NOT VALID WITHIN 'PROLYL?HYDROXYLASE'
The truncation symbol ? may be used only at the end of a search
term. To specify a variable character within a word use '!', e.g.,
'wom!n' to search for both 'woman' and 'women'. Enter "HELP
TRUNCATION" at an arrow prompt (=>) for more information.
```

```
=> S PROLYL ?HYDROXYLASE
    4465 PROLYL
    30651 ?HYDROXYLASE
L2    509 PROLYL ?HYDROXYLASE
        (PROLYL(W)?HYDROXYLASE)
```

```
=> S L1 (S) L2
L3    2 L1 (S) L2
```

```
=> S L1 (L) L2
L4    2 L1 (L) L2
```

```
=> D 1-2 IBIB ABS
```

```
L4    ANSWER 1 OF 2      MEDLINE
ACCESSION NUMBER: 2001548313      MEDLINE
DOCUMENT NUMBER: 21479120      PubMed ID: 11595184
TITLE: C. elegans EGL-9 and mammalian homologs define a family of
      dioxygenases that regulate HIF by prolyl hydroxylation.
COMMENT: Comment in: Cell. 2001 Oct 5;107(1):1-3
AUTHOR: Epstein A C; Gleadle J M; McNeill L A; Hewitson K S;
      O'Rourke J; Mole D R; Mukherji M; Metzen E; Wilson M I;
      Dhanda A; Tian Y M; Masson N; Hamilton D L; Jaakkola P;
      Barstead R; Hodgkin J; Maxwell P H; Pugh C W; Schofield C
      J; Ratcliffe P J
CORPORATE SOURCE: The Henry Wellcome Building of Genomic Medicine, Roosevelt
      Drive, Oxford OX3 7BN, United Kingdom.
SOURCE: CELL, (2001 Oct 5) 107 (1) 43-54.
      Journal code: 0413066. ISSN: 0092-8674.
PUB. COUNTRY: United States
      Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 20011015
      Last Updated on STN: 20020420
      Entered Medline: 20011204
```

```
AB    HIF is a transcriptional complex that plays a central role in mammalian
      oxygen homeostasis. Recent studies have defined posttranslational
      modification by prolyl hydroxylation as a key regulatory event that
      targets HIF-alpha subunits for proteasomal destruction via the von
      Hippel-Lindau ubiquitylation complex. Here, we define a conserved HIF-VHL-
      ***prolyl***      ***hydroxylase***      pathway in      ***C***
      ***elegans*** , and use a genetic approach to identify EGL-9 as a
      dioxygenase that regulates HIF by prolyl hydroxylation. In mammalian
```

series of isoforms bearing a conserved 2-histidine-1-carboxylate iron coordination motif at the catalytic site. Direct modulation of recombinant enzyme activity by graded hypoxia, iron chelation, and cobaltous ions mirrors the characteristics of HIF induction in vivo, fulfilling requirements for these enzymes being oxygen sensors that regulate HIF.

L4 ANSWER 2 OF 2 MEDLINE  
 ACCESSION NUMBER: 79021663 MEDLINE  
 DOCUMENT NUMBER: 79021663 PubMed ID: 212107  
 TITLE: In vitro translation of nematode cuticular collagens.  
 AUTHOR: Noble S; Leushner J; Pasternak J  
 SOURCE: BIOCHIMICA ET BIOPHYSICA ACTA, (1978 Aug 23) 520 (1) 219-28.  
 Journal code: 0217513. ISSN: 0006-3002.  
 PUB. COUNTRY: Netherlands  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 197812  
 ENTRY DATE: Entered STN: 19900314  
 Last updated on STN: 19900314  
 Entered Medline: 19781220

AB Phenanthroline treatment of growing cultures of the free-living \*\*\*nematode\*\*\* *Panagrellus silusiae* was used to lower the degree of hydroxylation of nascent collagen chains at the polysomal level. Under these conditions, the bound pentasome-hexasome fraction provided substrate for \*\*\*prolyl\*\*\* \*\*\*hydroxylase\*\*\*. When this polysomal fraction was subsequently tested in a cell-free wheat germ system, collagenase-susceptible translation products were observed after sodium dodecyl sulfate-acrylamide gel electrophoresis. The electrophoretic mobilities of each of these four major collagen products were similar to four collagens that are isolated from intact cuticles. In addition, purified polysomal RNA that adhered to unmodified cellulose directed the synthesis of four pepsin-resistant polypeptides that had molecular weights that coincided with four pepsin-resistant collagens that can be purified from the cuticle of this species. Thus, the polysomal site of the messenger RNAs for the cuticular collagens of *P. silusiae* was located. Although precursor forms of the cuticular collagens were not produced in the cell-free system, the question whether additional amino acid segments occur on the primary translational products of the cuticular collagens in vivo remains open.

=> D HIS

(FILE 'HOME' ENTERED AT 14:44:43 ON 18 JUL 2002)

FILE 'MEDLINE' ENTERED AT 14:44:53 ON 18 JUL 2002

L1 19214 S NEMATODE OR C.ELEGANS OR ?ELEGANS  
 L2 509 S PROLYL ?HYDROXYLASE  
 L3 2 S L1 (S) L2  
 L4 2 S L1 (L) L2

=> S DPY-18

103 DPY  
 365958 18  
 L5 7 DPY-18  
 (DPY(W)18)

=> S DPY-18 OR DPY

103 DPY  
 365958 18  
 7 DPY-18  
 (DPY(W)18)  
 103 DPY  
 L6 103 DPY-18 OR DPY

=> S L1 (L) L6

L7 59 L1 (L) L6

=> S L7 AND L2

L8 0 L7 AND L2

=> D HIS

(FILE 'HOME' ENTERED AT 14:44:43 ON 18 JUL 2002)